IN THE SPECIFICATION:

On page 7, line 16 - page 8, line 17, please amend the Specification as follow:

--FIG. 1 is a graph showing results of the cell based TNF neutralization assay with antibodies of the present invention compared to a control antibody. The L929 cell based assay shows the greater neutralization ability of anti-TNF IgY as compared to REMICADE. The dose of antibody which prevents 50 percent of the cytotoxicity associated with TNF (ND50) is 70 ng/ml for the anti-TNF IgY, and 140 ng/ml for REMICADE. Note the concentrations graphed for the REMICADE antibody represent the total amount of specific antibody, whereas the anti-TNF IgY concentrations represent total Igy Concentrations.

FIGS. 2A through 2D show results of a dose response study in a rat model for IBD using different concentrations of one embodiment of the anti-cytokine antibody of the present invention (i.e. anti-TNF IgY). Figure 2A shows the highest oral dose of anti-TNF prevented TNBS-induced animal weight loss as compared to the vehicle control and lower doses (*n=8, p,0.05 for all time points). The 7.5 mg/Day dose at days 6 and 10, and on day 7 was higher than the vehicle group (*n=8, p,0.05). Figure 2B shows oral treatment with anti-TNF reduced the mean total colon weight at all doses measured. The 120 mg/day dose is statistically significant as compared to the vehicle and lower doses (*n=8, p≤0.005, p≤.01). Figure 2C shows oral treatment with the highest dose of anti-TNF significantly reduced the histological damage as compared to the vehicle and two other doses (*n=8, p≤.0025, p≤0.005, p≤.025). Figure 2D shows oral administration of 120 mg/day of anti-TNF significantly reduces MPO levels as compared to the vehicle control, as well as the 7.5 mg/day and 30 mg/day doses.

FIGS. 3A through 3D are bar graphs showing the results after pre-challenge treatment with vehicle, preimmune IgY, or an anti-TNF IgY antibody of the present invention. Figure 3A shows oral delivery of anti-TNF antibodies reduced colonic weight gain associated with TNBS treatment. The anti-TNF reduction was statistically significant compared to the preimmune treated control (*n=8, p≤0.001). Figure 3B shows oral delivery of anti-TNF reduced histological damage for rats treated with TNBS as compared to vehicle (n=7, *p,0.05) and preimmune controls (N=8, *p≤0.001). Normal control was zero. Figure 3C shows treatment with anti-TNF reduces histological damage as compared to the vehicle and preimmune control animals (*n=7,

$p \leq 0.05$, and $p \leq 0.0025$ respectively). Normal control was zero. Figure 3D shows oral delivery of anti-TNF antibodies significantly reduced the levels of tissue mysloperoxidase after rat treatment with TNBS as compared to the preimmune (*n=7, $p \leq 0.05$).

FIGS. 4A through 4D are bar graphs showing the results after post-challenge (48 hours) treatment with vehicle, sulfasalazine, or an anti-TNF IgY antibody of the present invention. Figure 4A shows oral administration of anti-TNF antibodies to rats 48 hours post treatment with TNBS significantly decreased disease associated colonic weight gain as compared to vehicle treated animals (*$p \leq 0.05$), saw no improvement over controls. Figure 4B shows oral administration of anti-TNF antibodies to rats 48 hours post treatment with TNBS significantly decreased morphological damage as compared to vehicle control (n=7, * $p \leq 0.005$). Figure 4C shows treatment with anti-TNF significantly reduces histological damage as compared to the sulfasalazine and vehicle treated controls (*n=7, $p \leq 0.01$ and $p \leq 0.001$ respectively). Figure 4D shows oral administration of anti-TNF antibodies to rats 48 hours post TNBS treatment significantly reduced the tissue mysloperoxidase as compared to vehicle (n=7, *$p \leq 0.002$) and sulfasalazine (n=7, *$p \leq 0.02$).

FIGS. 5A through 5D are bar graphs showing the results after pre-challenge treatment with vehicle, dexamethasone, or an anti-TNF IgY antibody of the present invention. Figure 5A shows oral administration of anti-TNF and dexamethasone significantly reduce total colon weight as compared to the vehicle control (n=9, *$p \leq 0.025$ and $p \leq 0.05$ respectively). Figure 5B shows oral administration with anti-TNF significantly reduces morophological damage as compared to the vehicle and dexamethasone treated groups (*n=9, $p \leq 0.05$ and $p \leq 0.025$ respectively). Figure 5C shows oral administration with anti-TNF significantly reduces histological damage as cmopared to the dexamethasone and vehicle treated animals (*n=9, $p \leq 0.001$ and $p \leq 0.01$). Figure 5D shows oral administration of anti-TNF significantly reduces mysloperoxidase levels as compared to dexamethasone treated animals (*n=9, $p \leq 0.05$).

FIGS. 6A through 6C are bar graphs showing the results of anti-TNF treatment started 17 days after initiation of an inflammatory reaction in a rat model for IBD. Figure 6A shows oral treatment with anti-TNF significantly decreases the colon weight as compared to the vehicle and preimmune controls (*$p \leq 0.025$ vs. vehicle and preimmune). Figure 6B shows oral treatment with anti-TNF significantly decreases morphological damage as compared to the vehicle and

preimmune controls (*p≤0.025 vs. vehicle and preimmune). Figure 6C shows oral treatment with anti-TNF significantly decreases the microscopic damage as compared to vehicle and preimmune controls (p≤0.025 and p≤0.01 respectively).

FIG. 7 is a bar graph showing MPO activity in colons of anti-TNF treated mice and survivors of preimmune and vehicle treated mice. Intrarectal delivery of anti-TNF antibodies beginning days 3 of a 7 day DSS treatment regimen significantly reduces tissue myeloperoxidase levels as compared to preimmune control (n=5, *p≤0.05), but not the vehicle control (n=10, *p≤0.2).

FIGS. 8A through 8C are bar graphs showing the treatment results in a mouse model (C3H/HeJ mice) of IBD involving treatment rectally with anti-TNF, vehicle, or preimmune immediately following a single five day cycle of 5% DSS. Figure 8A shows intrarectal delivery with anti-TNF after 5 days of DSS treatment significantly reduced ocult blood found in the stool of DSS treated mice as compared to the vehicle control (*p≤0.05). Figure 8B shows intrarectal treatment with anti-TNF antibodies after 5 days of DSS treatment significantly reduced histological damage as compared to vehicle control (*p≤0.02). Figure 8C shows intrarectal delivery of anti-TNF antibodies after 5 days of DSS treatment decreased tissue myeloperoxidase levels as compared to the vehicle control (*p≤0.05) and the preimmune control (*p≤0.05).